entirely through the osseous structures, causing the splitting fracture of the posterior elements, but in this instance there is sparing of the ligamentous structures. The fracture usually extends horizontally from the tip of the transverse process on one side through the pedicles and laminae to the tip of the opposite transverse process. It may extend into the posterior aspect of the vertebral body, and occasionally there may be avulsion of one or both of the superior articular processes. Involvement of the anterior aspect of the body is not a constant feature, and when it does occur it is usually not severe.3-7

Visceral injuries may be associated with either kind of fracture, but neurologic deficits are uncommon, since major subluxation is ordinarily not a feature of this syndrome.

In the case herein reported, both ligamentous and osseous elements were involved-a horizontal splitting fracture and rupture of the posterior ligamentous structures. This is the first known instance of this combination.

Summary

Seat belt fracture in the case herein reported resulted from wearing the seat belt high over the abdomen at the level of the umbilicus rather than over the pelvis. In injuries of this kind, at the time of impact the belt acts as a fulcrum around which the body pivots, directing the major vector of force toward the lumbar spine. This may result in one of several injury patterns. There may be gross separation of the posterior elements due to rupture of the posterior supporting ligamentous structures, or the force may be entirely absorbed by the osseous structures. In the latter instance there is a horizontal splitting fracture of the neural arch which extends through the lamina and pedicles to involve both transverse processes.

REFERENCES

- 1. Tourin B, Garrett JW: Seat Belt Effectiveness in Rural California Automobile Accidents: A Comparison of Injuries to Users and Nonusers of Safety Belts. Automotive Crash Injury Research of Cornell University, New York, Feb 1970
- 2. Smith WS, Kaufer H: Patterns and mechanisms of lumbar in-tries associated with lap seat belts. J Bone Joint Surg 51-A:239-
- 3. Carroll TB, Gruber FH: Seat belt fractures. Radiology 91:517-518, 1968
- 4. Chance GQ: Note on a type of flexion fracture of the spine. Brit J Radio 21:452-453, 1948
- 5. Garrett JW, Braunstein PW: The seat belt syndrome. J Trauma 2:220-238, 1962
- 6. Howland WJ, Curry JL, Buffington CB: Fulcrum fractures of the lumbar spine: Transverse fracture induced by an improperly placed seat belt. JAMA 193:240-241, 1965
- 7. Stechler RM, Epstein J, Epstein BS: Seat belt trauma to the lumbar spine. JAMA 207:758-759, 1969

Primary Invasion by Clostridium Sphenoides In a Patient with Periodic Neutropenia

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PRIMARY INVASION OF HEALTHY undamaged tissue by any of the pathogenic Clostridia is an improbable event. The presence of necrotic tissue is a prerequisite for the establishment of a focus of anaerobic infection. Primary invasion by a nonpathogenic species of Clostridium is almost unique. The case to be described represents the first reported instance of Clostridium sphenoides infection in a human being. It involved the nontraumatized colon of a patient with periodic neutropenia and led to rapid death.

The patient was a 6-year-old Eurasian girl who had a lifelong history of recurrent attacks of otitis media, oral ulcers, periodontal abscesses and chronic gingivitis. At age 2 she was found to have neutropenia, which was persistent but, as determined by specific investigation, was not of rhythmic pattern. In a total of 21 leukocyte differential determinations in her lifetime, the neutrophil count was within normal limits on only one occasion, not associated with an episode of infection. (Chart 1). Occasionally atypical lymphocytes were noted. Platelets were normal and the hematocrit ranged from 31 to 40 percent, usually being in the mid 30's. Bone marrow examination during the neutropenia showed a delay and partial arrest in granulocytic maturation. The gamma globulin level was normal. Results of repeated urinalysis were within normal limits.

Because of the high frequency of the patterns of infection described above, the patient often received antibiotic therapy, and once for a period of a year she received erythromycin in prophylactic dosage. Coagulase positive Staphylococcus

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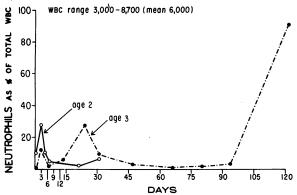


Chart 1.—Results of serial differential leukocyte counts at age 2 and 3.

aureus was cultured repeatedly from oral abscesses. All the patient's teeth had been extracted to gain control of these infections. In general no mode of therapy was clearly successful. The patient's growth, however, was appropriate for her age.

A sister had a history of similar, though less frequent, oral infections. A brother and the parents are asymptomatic. It has not been possible to study any of them.

At age 6, during a minor local outbreak of presumed viral gastroenteritis, the patient had abdominal cramps, occasional vomiting and an initial fever of 39.4°C (103°F). When seen approximately 20 hours after onset she was afebrile, somewhat listless and chronically ill in appearance. She was not dehydrated. Physical examination was normal except for the abdomen. There was no guarding or localized tenderness on palpation. Peristaltic rushes were occasionally felt. Bowel sounds were erratic and slightly decreased and borborygmi were heard. None of the organs was enlarged.

The history of neutropenia was not obtainable from the parents and she was treated with an antispasmodic agent, with moderate initial response. When the child was next seen nine hours later, the abdomen was rigid and bowel sounds were absent. Death occurred several minutes later.

A complete autopsy* was performed eight hours after death. Abnormalities were limited to the abdomen. The distal half of the ascending colon was inflamed and hemorrhagic with multiple small gas blebs both in the wall and in the

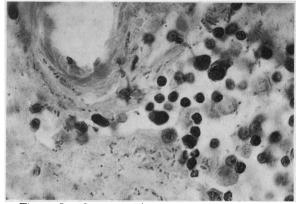


Figure 1.—Section of colon showing numerous organisms with neutrophil-free inflammatory response (X960).

adjacent mesenteric lymph nodes. No ulcerations were found and no areas of perforation could be identified. The appendix was normal grossly and microscopically. Histologic examination of the involved colon wall showed large numbers of Gram-positive spore forming rods. The majority of the inflammatory cells present were mononuclear, with polymorphonuclears being conspicuously absent (Figure 1). The blood was sterile. Culture of material from the peritoneum grew pure Clostridium sphenoides.

Cultural characteristics of this organism were: growth at 37°C, obligate anaerobe with spherical, subterminal spores. Colonies were hemolytic with irregular edges. The organism was non-virulent for mice. Gelatin was not liquefied but litmus milk, glucose, maltose, lactose, sucrose, mannitol, and salicin all gave acid reactions. The organism was motile, produced indole, and reduced nitrates. It gave negative reaction to urea and H₂S tests.

Discussion

This case appears to be the first clearly documented instance of infection by C. sphenoides in a human being. MacLennan¹ isolated C. sphenoides from war wounds in the North Africa campaign of World War II, but it is clear from his reports that he considered it a contaminant. The one case of anaerobic cellulitis from which he isolated C. sphenoides contained C. welchii and C. sporogenes. C. sphenoides was also cultured from 6 of 146 cases of anaerobic myositis, but again only in the presence of standard pathogens like C. welchii.

^{*}Captain Kent Smith, U.S. Army Medical Corps, performed the autopsy and supplied the culture results. The organism was identified by the 1st U.S. Army Medical Laboratory, Ft. Meade, Md.

In the original report of the discovery of Clostridium sphenoides² the medical aspects were referred to in unpublished data and it is not possible to tell whether the three patients from whom the organism was isolated in pure culture had gas gangrene or whether simple wound surface colonization was being reported. We have not been able to locate any report of solitary C. sphenoides infection. Indeed the organism is very uncommon in human feces³ and has been found in only 4 percent of soil samples.4

Another unusual aspect of this case is that it represents primary invasion of apparently healthy tissue. Ordinarily the absolute prerequisite for clostridial infection is a focus of necrotic tissue which is then invaded by this organism.⁵ Reports of invasion without an evident necrotic focus (or a probable one as in a cancer) are quite rare. 6,7,8,9 Enteritis necrotans, or darmbrand, once thought to represent primary invasion, is now known to be a toxic necrosis with secondary invasion by type F C. welchii.10 Other members of the patient's family, eating as a unit, were not ill.

Any explanation of the apparent primary invasion here, to be in keeping with standard knowledge, 11 would have to involve an undetected microscopic area of anoxia in the wall of the ascending colon. Quite reasonably an area of edematous ischemia related to the clinical diagnosis of gastroenteritis, or a microabscess caused by another organism, would provide an adequate foothold. One is led to conclude, by the rare combination of events, that the patient's inability

to mount a neutrophilic response was a major factor in permitting the dissemination of the initial focus.

The last point of interest in this case is that it indicates that periodic neutropenia can be a life threatening condition. In most of the cases described by Reimann¹² the patients survived to adulthood. The likelihood of survival would seem to be related to how much of the time, and to what degree, a given patient is neutropenic.

Summary

The first documented case of Clostridium sphenoides infection of a human being is described. This was an instance of apparent primary invasion by the organism in a patient with periodic neutropenia, leading to rapid death.

REFERENCES

- MacLennan JD: Anaerobic infections of war wounds in the Middle East. Lancet 2:94, 1943
- 2. Report of the Committee upon Anaerobic Bacteria and Infections.' Medical Research Council Special Report Series, Great Britain, #39, 1919
- 3. Kahn MC: Anaerobic spore bearing bacteria of the human intestine in health and in certain diseases. J Infect Dis 35:423, 1924
- 4. Lindberg RB: The bacterial flora of battle wounds at the time of the primary debridement. Ann Surg 141:369, 1955
- 5. MacLennan JD: Anaerobic infections of war wounds in the Middle East. Lancet 2:63, 1943
- 6. Cone SM: Osteomyelitis at the sacro-iliac joint with gas bacillus infection. Am J Orthoped Surg 11:389, 1913
- 7. Kimball HW, Rawson AJ: Non-traumatic gas gangrene. Virginia Med Monthly 79:269, 1952
- 8. Killingback MJ, Williams KL: Necrotizing colitis. Brit J Surg 49:175, 1961
- 9. Schoenfeld MR, Messeloff CR, Lepow H: Clostridium botulinum isolated from ascitic fluid. Am J Gastroent 37:537, 1962
- 10. Zeissler J. Rassfeld-Sternberg L: Enteritis necrotans due to C. welchii, Type F. Brit Med J 1:267, 1949
- 11. MacLennan JD: The histotoxic clostridial infections of man. Bacteriol Rev 26:177, 1962
- 12. Reimann HA: Periodic Diseases. Philadelphia, FA Davis Co, 1963

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